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Understanding chronic ankle instability: model rich, data poor

Chris Bleakley ¹, Jente Wagemans ², Fredh Netterström-Wedin³

Ankle sprains are common sports injuries. Although often perceived as innocuous, large proportions of patients develop a cluster of persistent symptoms, termed chronic ankle instability (CAI). In 1965, Freeman¹ first described a clinical paradox whereby poor recovery after ankle sprain (characterised by feelings of ‘giving way’) was reported in both the presence and absence of mechanical instability (MI). Since then, researchers have tried to explain this anomaly using original research, theoretical frameworks and multicomponent aetiological models for CAI. In this editorial, we discuss perennial and fundamental shortcomings in the evidence base, that continue to limit our understanding of CAI causation.

EVER-INCREASING COMPLEXITY

Most original research examining CAI aetiology is case-controlled. While this is both practical and cost-saving, its data can only ‘explain’ CAI aetiology after the fact. Few studies have prospectively tracked recovery post ankle sprain,^{2,3} with even fewer prognostic factors emerging. As a result, the content of aetiological models in this field^{1,4,5} is either theoretical or driven primarily by cross-sectional data. An inevitable consequence is an ever-increasing model complexity, with each iteration adding additional clinical constructs. This posits a trend towards a more phenomenological and multicausal understanding of CAI, which has cumulated in a flexible aetiological model that can accommodate virtually any pattern of observed data. However, in the absence of prospective data or validation research, it is still unclear which model variables are causative, associative or by-products

of the condition, making causal control and clinical application difficult.

LATENT INSTABILITIES

Cross-sectional data seem to corroborate Freeman’s original observation, with reports that 76% of patients with CAI do not present with mechanically unstable ankles.⁴ A popular supposition is that this pattern is due to sprain-induced deafferentation or damage to ligament mechanoreceptors (without any alteration to the ligament’s biomechanical properties). Consequently, research examining MI, or passive stability of the ankle joint, has diminished in recent years, with researchers focusing more on the dynamic components of sensorimotor control, or patient-reported outcomes.

But, prospective research examining MI as a primary causative factor of CAI is both limited and equivocal.^{2,3} Perhaps the most fundamental limitation is that, in the majority of research studies, MI is assessed by stress-testing a single ankle ligament (usually the anterior talofibular ligament, ATFL). Although the ATFL is the most injured ankle ligament, testing it in isolation lacks basic face validity, as it constitutes a small proportion of the connective tissue that stabilises the foot and ankle complex. Indeed, the joints in this region (syndesmosis, talocrural and subtalar joint) are anatomically and functionally inseparable and are stabilised by several ligaments (eg, anterior inferior tibio fibular; interosseous talocalcaneal; cervical) that are prone to both isolated and combined ligamentous injury during ankle torsion.

An obvious solution for assessing MI is to extend the clinical examination beyond the ATFL, but few tests are accurate and valid. Although the talar tilt test evaluating the calcaneofibular ligament has excellent specificity (95% CI 84% to 94%), it has low sensitivity (95% CI 12% to 23%).⁶ The more fundamental problem is that the test is often interpreted based on pain replication rather than joint stability. Similarly, clinical assessment of the ankle syndesmosis is also based on pain provocation, and accurate prediction of joint instability

necessitates a range of diagnostic procedures including arthroscopy;⁷ examinations rarely employed in the CAI literature. Subtalar instability can occur in conjunction with talocrural instability, but clinical examination alone cannot differentiate these conditions. The term ‘microinstability’ is increasingly used in contemporary surgical literature and represents ligament laxity that is only evident through arthroscopy. Disappointingly, it seems that even the most tried and tested of clinical stress test (anterior drawer), does not consistently detect microinstabilities involving the ATFL.⁸

THE FUTURE

Existing models of CAI aetiology are comprehensive, but they represent open concepts: defined and classified within a complex symptom-based framework. If current research trends persist (ie, high volume of cross-sectional data; lack of validation studies), CAI frameworks will, inevitably, develop further into compound representations of symptom clusters, without an identifiable aetiology. MI remains as the most likely intervening variable, which is a consequence of ankle trauma and a causal component of CAI; yet this has not been rigorously and validly examined through prospective research. New anatomical data continue to emerge (eg, the intra-articular nature of the ATFL’s superior fascicle, or the arcuate nature of the lateral fibulotalocalcaneal ligament complex),⁸ which should also challenge our diagnostic and treatment dogma. Future studies must evolve beyond a perfunctory assessment of MI to consider the stability of all primary ligaments in the foot and ankle: using a combination of clinical assessment, medical imaging and arthroscopy.

There has been much investment and academic interest in this field, but if we set the bar high, it seems that Freeman’s paradox—first presented over 50 years ago, remains largely unsolved.

Twitter Jente Wagemans @JenteWagemans

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¹Health Sciences, Faculty of Life and Health Sciences, Ulster University, Newtownabbey, UK

²Department of Rehabilitation Science and Physiotherapy, University of Antwerp Faculty of Medicine and Health Sciences, Antwerp, Belgium

³Department of Community Medicine and Rehabilitation, Umea Universitet, Umea, Sweden

Correspondence to Dr Chris Bleakley, Health Sciences, Ulster University Faculty of Life and Health Sciences, Newtownabbey, UK, BT370QB; chrisbleakley77@gmail.com

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ORCID iDs

Chris Bleakley <http://orcid.org/0000-0001-9032-9691>

Jente Wagemans <http://orcid.org/0000-0001-7856-463X>

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